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CASE REPORT

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Near-infrared spectroscopy as a predictor of clinical deterioration: a case report of two infants with duct-dependent congenital heart disease

Mirthe J. Mebius^{1*}, Gideon J. du Marchie Sarvaas³, Diana W. Wolthuis², Beatrijs Bartelds³, Martin C. J. Kneyber^{2,4}, Arend F. Bos¹ and Elisabeth M. W. Kooi¹

Abstract

Background: Some infants with congenital heart disease are at risk of in-hospital cardiac arrest. To better foresee cardiac arrest in infants with congenital heart disease, it might be useful to continuously assess end-organ perfusion. Near-infrared spectroscopy is a non-invasive method to continuously assess multisite regional tissue oxygen saturation.

Case presentation: We report on two infants with duct-dependent congenital heart disease who demonstrated a gradual change in cerebral and/or renal tissue oxygen saturation before cardiopulmonary resuscitation was required. In both cases, other clinical parameters such as heart rate, arterial oxygen saturation and blood pressure did not indicate that deterioration was imminent.

Conclusions: These two cases demonstrate that near-infrared spectroscopy might contribute to detecting a deteriorating clinical condition and might therefore be helpful in averting cardiopulmonary collapse and need for resuscitation in infants with congenital heart disease.

Keywords: Congenital heart disease, Near-infrared spectroscopy, Newborn infants, Cardiac arrest, Cardiopulmonary resuscitation, Intensive care, Case report

Background

Some infants with congenital heart disease (CHD) are at risk of cardiac arrest during hospitalization [1]. In-hospital cardiac arrest occurs in approximately 2% to 6% of children admitted to paediatric intensive care units [2, 3]. Through earlier recognition, cardiac arrest may be prevented and outcomes and survival may be improved [4].

Monitoring end-organ perfusion might help recognizing clinical deterioration at an early stage. Near-infrared spectroscopy (NIRS) is a non-invasive clinical tool to continuously assess multisite regional tissue oxygen saturation (rSO₂) [5]. It is based on the relative transparency of

biological young tissue (bone, skin, soft tissue) and the ability to differentiate oxygenated hemoglobin from deoxygenated hemoglobin as they have distinct absorption spectra. The ratio between oxygenated and total hemoglobin represents the regional tissue oxygen saturation [6]. In infants with CHD, multiple studies have investigated tissue oxygenation at various moments during early life [7–14]. Most studies addressed the intraoperative and postoperative period [7–10], but cerebral rSO₂ was also assessed preoperatively in some studies [11–14]. To our knowledge, however, multisite tissue oxygen saturation as a predictor of clinical deterioration in neonates with CHD has not been described before.

We discuss two cases in which continuous monitoring of multisite tissue oxygen saturation using NIRS indicated a deteriorating clinical condition which eventually led to cardiac arrest.

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Case presentation

Both cases in this report were part of a prospective observational cohort study (registration number NTR5233), which was conducted between May 2014 and August 2016. Parental informed consent was obtained in both cases. We measured cerebral and renal rSO₂ using INVOS 5100c near-infrared spectrometers (Somanetics Corporation, Troy, Michigan, USA) in combination with neonatal sensors (Somanetics Corporation). Nurses were not instructed to act on certain rSO₂ values, as they were not measured for clinical purposes.

Case 1

A boy diagnosed prenatally with pulmonary atresia with an intact ventricular septum was born at our hospital at a gestational age of 37.3 weeks. After birth, the patient was admitted to the neonatal intensive care unit (NICU) with continuous positive airway pressure (CPAP) at seven cmH₂O with a maximum of 40% O₂ and infusion with prostaglandin E₁ (0.025 ug/kg/min) to maintain pulmonary circulation. Prenatal diagnosis and ductal patency were confirmed by the pediatric cardiologist. The patient appeared to be hemodynamically stable with transcutaneous oxygen saturations (SpO₂) between 70% and 90% and mean arterial blood pressures between 46 and 73 mmHg. As the patient was clinically stable, further hemodynamic parameters were not available for this case.

Four hours after birth the boy developed three apneas. The third apnea was followed by loss of cardiac output. Cardiopulmonary resuscitation was initiated immediately according to protocol. Cardiac output and heart rate, however, remained insufficient and the boy died 6 h after birth.

Autopsy confirmed the diagnosis of a pulmonary atresia with a hypoplastic right ventricle and an intact

ventricular septum. Furthermore, autopsy revealed a single coronary ostium, multiple ventricular-coronary fistulas and a right ventricle dependent coronary blood flow with bridging of a branch of the right coronary artery. There were signs of recent myocardial ischemia, such as loss of cardiomyocytes. In addition, there was a left-sided open ductus arteriosus between the brachiocephalic trunk and the pulmonary artery. Based on autopsy findings, the most likely cause of death was considered to be massive myocardial ischemia which is not uncommon for this type of anatomy and physiology. Other causes such as a primary intraventricular hemorrhage or infarction cannot be ruled out completely since there was no permission for cerebral autopsy.

Near-infrared spectroscopy case 1

Baseline cerebral and renal rSO₂ were relatively low (around 40%) and decreased rapidly approximately 40 min before cardiopulmonary resuscitation was necessary (Fig. 1). This rapid decrease in cerebral and renal rSO₂ was probably due to a sudden decrease in arterial oxygen saturation. All clinical parameters spontaneously recovered from this incident. Cerebral rSO₂, however, started decreasing again from approximately 40% to 15% 25 min before cardiopulmonary resuscitation was necessary, while other hemodynamic parameters did not alarm the attending staff. Within 10 min, cerebral rSO₂ had already decreased 50% from its original baseline value (40%) to approximately 20%. We cannot fully explain why cerebral rSO₂ decreased before renal rSO₂ and SpO₂.

Case 2

A girl diagnosed prenatally with double outlet right ventricle, transposed great arteries, a ventricular septal defect and a hypoplastic aortic arch was born prematurely at our hospital at a gestational age of 34.9 weeks.

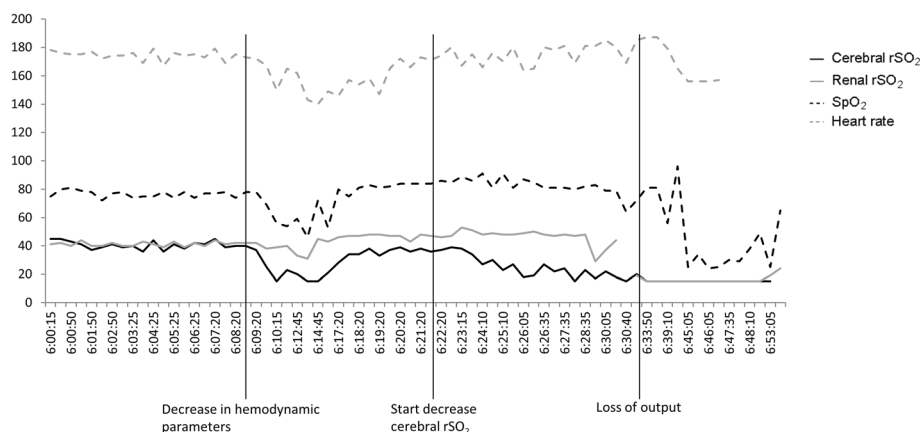


Fig. 1 The course of hemodynamic parameters and cerebral and renal oxygen saturation of the first case. SpO₂, transcutaneous arterial oxygen saturation; rSO₂, regional tissue oxygen saturation

She was admitted to the NICU with CPAP 10cmH₂O with a maximum of 40% O₂ due to respiratory distress/delayed transition. Initially, she had a wide-open ductus arteriosus. Eighteen hours after birth her clinical condition deteriorated with signs of a duct-dependent systemic circulation. Therefore, prostaglandin E₁ infusion was administered (0.025 ug/kg/min) to maintain ductal patency. During the first days after birth there was a fragile balance between pulmonary and systemic blood flow.

At the sixth day after birth, she underwent bilateral pulmonary banding with the intention of controlling an ever increasing pulmonary blood flow. Peri-operatively, hypotension was treated successfully with multiple volume expansions and noradrenalin. Postoperatively, she was on Bi-level Positive Airway Pressure (BiPAP)-assist and prior to loss of output there were no clinical signs of primary respiratory failure (e.g. inadvertent extubation). Arterial pH was 7.34, arterial saturation was 90%, pCO₂ was 4.8 kPa, hemoglobin was 8.0 mmol/l and lactate was 1.5 mmol/l. At that time, arterial blood pressure was 70/30 mmHg and she had normal diuresis. One hour later, she suddenly developed bradycardia followed by loss of cardiac output. Cardiopulmonary resuscitation was initiated immediately according to protocol. Infusion with prostaglandin E₁ was transferred from a peripheral venous site to a central line and was increased in dosage. An echocardiogram suggested an open ductus arteriosus, revealed a poor contractility and an inadequate cardiac output. After 40 min of cardiopulmonary resuscitation, the sternum was re-opened. The heart, however, was almost non-contractile and the girl died 7 days after birth.

Autopsy confirmed the diagnosis and concluded that the lumen of the ductus arteriosus was small with a diameter of less than three millimeter. Whether this was too small to maintain systemic circulation and whether

prostaglandin E₁ infusion was inadequate at the time of collapse, remains unknown. Furthermore, autopsy revealed an immature lung parenchyma, atelectasis of part of the basal lobes and small inflammatory areas in both lungs. These pulmonary findings might also have contributed to the cardiopulmonary collapse.

Near-infrared spectroscopy case 2

We observed an increase in cerebral rSO₂ and a decrease in renal rSO₂ 1 h before loss of output (Fig. 2). Renal rSO₂ returned to baseline, while cerebral rSO₂ remained approximately 10% above baseline. This incident might have been caused by a closing ductus arteriosus due to insufficient Prostaglandin E₁ administration or an insufficient response to adequate Prostaglandin E₁ administration. Especially the rapidly decreasing renal rSO₂ was indicative of a closing ductus arteriosus. Due to the coarctation of the aorta, renal blood flow was dependent on an open ductus arteriosus. The increase in cerebral rSO₂ was less easy to explain, but could be caused by an increase in cerebral blood flow. Due to the closing ductus arteriosus, the descending aorta might have become narrower, leading to an increased arterial pressure above the coarctation and subsequently increased cerebral blood flow. Thirty minutes before cardiopulmonary resuscitation, cerebral rSO₂ started decreasing from 55% to approximately 20% and renal rSO₂ decreased from 40% to 15%. Renal rSO₂ decreased more rapidly compared with cerebral rSO₂ and approximately 15 min before loss of output, renal rSO₂ was less than 50% from its original baseline value. The more rapid decrease of renal rSO₂ in comparison with cerebral rSO₂ also supports the theory of insufficient systemic circulation due to a closing ductus arteriosus. Apart from a slowly increasing heart rate from 160 bpm to 180 bpm, hemodynamic parameters such as

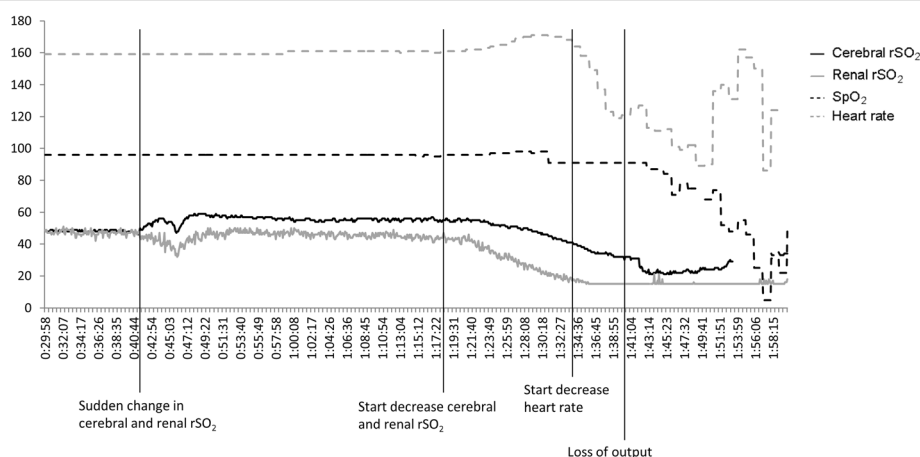


Fig. 2 The course of hemodynamic parameters and cerebral and renal oxygen saturation of the second case. SpO₂, transcutaneous arterial oxygen saturation; rSO₂, regional tissue oxygen saturation

SpO₂ and arterial blood pressure remained stable until several minutes before cardiopulmonary resuscitation was necessary (Fig. 2).

Discussion

These cases demonstrate that continuous monitoring of multisite near-infrared spectroscopy might be helpful in early identification of clinical deterioration in infants with congenital heart disease. In both cases, cerebral and/or renal rSO₂ changed 30 min or more before loss of output while other hemodynamic parameters did not indicate that deterioration was imminent until a few minutes before cardiopulmonary resuscitation was necessary.

Cerebral rSO₂ decreased 30–35% from baseline before loss of output. McNeill et al. found that cerebral rSO₂ values varied 15% or more from baseline rSO₂ values during less than 1% of the day in preterm infants [15]. Furthermore, Bernal et al. demonstrated an average cerebral rSO₂ of 77% with a standard deviation of 6.1% within patients during the first week after birth [16]. As a decrease of 30–35% exceeds this intra-individual variability, it seems more likely that the decrease in cerebral rSO₂ observed in the two cases was a consequence of a deteriorating clinical condition and a subsequent reduction of cerebral perfusion, rather than the normal intra-individual variability of NIRS.

In the current cases we did not respond to changes in cerebral and renal rSO₂, as they were measured in the context of a prospective observational cohort study and not for clinical purposes. In retrospect, these cases indicate that large changes in cerebral and/or renal rSO₂ in infants with duct-dependent CHD should be considered a red flag and warrant a complete check-up of the infant, including physical examination, echocardiography and basic blood tests. The time-interval of 15 to 30 min might give some extra time to prevent clinical deterioration in infants with CHD. Potential therapeutic strategies depend on the reason of the change in cerebral and/or renal rSO₂, but could include mechanical ventilation, pharmaceutical therapy (e.g. inotropes), volume expansion or perhaps emergency surgery. Furthermore, it might be useful to use predefined upper and lower limits of cerebral and renal oxygen saturation, a principle that was recently suggested in a large trial with preterm infants [17]. Due to the heterogeneity of the cardiac population, it is difficult to apply normal ranges of cerebral and renal rSO₂. In this population it might be more useful to use limits below which histological and biochemical changes in tissue have been reported, i.e. below 44% [18].

Conclusions

In conclusion, in infants with duct-dependent congenital heart disease, a significant change in cerebral and/or renal oxygen saturation from baseline as measured by

NIRS may be an early indicator of a sudden, unexpected clinical deterioration, which eventually could lead to cardiac arrest and death. Increased awareness and thorough scrutiny of the patient are required when such changes in cerebral and/or renal oxygen saturation are observed.

Abbreviations

BiPAP-assist: Bi-level Positive Airway Pressure - assist; CHD: Congenital heart disease; CPAP: Continuous positive airway pressure; NICU: Neonatal intensive care unit; NIRS: Near-infrared spectroscopy; rSO₂: Regional tissue oxygen saturation; SpO₂: Transcutaneous arterial oxygen saturation

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Availability of data and materials

The datasets during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Authors' contributions

MJM made substantial contributions to the conception and design of the study, drafted the initial manuscript, revised the manuscript after feedback from co-authors, and approved the final manuscript as submitted. GJdMS was one of the pediatric cardiologists involved in the second case, made substantial contributions to the interpretation of data, critically reviewed the manuscript, and approved the final manuscript as submitted. DWW was the attending staff member in the second case, made substantial contributions to acquisition of data, critically reviewed the manuscript, and approved the final manuscript as submitted. BB was one of the pediatric cardiologists involved in the first case, made substantial contributions to acquisition of data, critically reviewed the manuscript, and approved the final manuscript as submitted. MCJK made substantial contributions to the interpretation of data, critically reviewed the manuscript, and approved the final manuscript as submitted. AFB made substantial contributions to the conception and design of the study, critically reviewed the manuscript, and approved the final manuscript as submitted. EMWK was the attending staff member in the first case, made substantial contributions to the conception and design of the study and the interpretation of data, critically reviewed the manuscript, and approved the final manuscript as submitted.

Authors' information

Not applicable

Competing interests

The authors declare that they have no competing interests.

Consent for publication

In both cases we received written informed consent from both parents to publish the information in this case report.

Ethics approval and consent to participate

The Medical Ethics Review Board University Medical Center Groningen¹ approved the prospective observational cohort study and parental informed consent was obtained before inclusion in the study.

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